

2011

The Not-so-Sweet Side of Sugars

Maximilian Klein
New York Medical College

Follow this and additional works at: https://touro scholar.touro.edu/quill_and_scope



Part of the [Arts and Humanities Commons](#), [Higher Education Commons](#), and the [Medicine and Health Sciences Commons](#)

Recommended Citation

Klein, M. (2011). The Not-so-Sweet Side of Sugars. *Quill & Scope*, 4 (1). Retrieved from

This Review is brought to you for free and open access by the Students at Touro Scholar. It has been accepted for inclusion in Quill & Scope by an authorized editor of Touro Scholar. . For more information, please contact touro.scholar@touro.edu.



The Not-so-Sweet Side of Sugars

Maximilian Klein

Medical school is funny sometimes. Every now and then in lecture I hear the professor say something that makes me think, "Why was I not told this before" In this light, I feel obligated to expand on one particular experience for the benefit of our population.

I was caught off guard the day we covered fructose metabolism in a series of lectures on carbohydrate biochemistry when my professor made the comment, "At one time, fructose was experimentally tested to see if it could be used as a substitute for glucose. It turned out to be very bad for the patients."

Very bad for the patients? When did they figure this out? I made sure to see my professor after class to ask him more about this, and he casually told me that scientists have known about the harmful effects in humans of high fructose feeding for quite some time.^{1,2} I found out that if you force feed fructose into a mouse it will develop diabetes and diabetes related conditions³⁻⁶, or as current journals like to put it, "deleterious metabolic effects".⁷

So often, media health correspondents and public health officials makes hints to stay away from high fructose corn syrup (HFCS), but has anyone ever explained *why* the public should stay away from large amounts of fructose? Or better yet, why the sugar fructose is particularly bad for our health when compared to other dietary components? As a public health student as well as a medical student I think the science needs to be better communicated from the research bench to the public with more understandable explanations and fewer "just do as I say" rules.

First, I should start out with a little refresher on the basics of sugars. They take on many names and forms but essentially they are all cousins to one another; that is, they are all related to each other by their chemical structure. Two common sugars that we have all heard of are sucrose (table sugar) and lactose (found in dairy products). Sucrose and lactose are what we call disaccharides, meaning that they have two smaller sugar molecules linked together in a chemical bond. In order to transport the sucrose and lactose from our food into our blood stream we have to first break down the disaccharides into their individual units. If we do not break the disaccharides into individual monosaccharides, the bacteria downstream in our intestines quickly do it for us and release copious gas as a by-product, hence the potentially uncomfortable condition *lactose intolerance*, which basically results from an individual's inability to break down lactose into its individual sugar units.⁸ Ok, let's get back to the main story.

The two units that make up sucrose are glucose and *fructose*. Ah-hah! Sucrose is a 50/50 ratio of glucose and fructose. So what is the big deal about HFCS since the commercial form is usually 55% fructose and 45% glucose? Well if you will take my word for now that fructose is dangerous, then HFCS has a 5% increase in danger which theoretically is less good. But back to the central question, "Why is fructose so bad?" To answer this question the science behind how fructose is metabolized needs to be explored.

“
...has anyone ever explained why the public should stay away from large amounts of fructose?”

Let us consider glucose first. When glucose enters your bloodstream (whether from the 50% component of your table sugar, your morning cereal, or your sandwich bread) your blood sugar (i.e. blood glucose) will raise above the normal, healthy level. The raise in blood sugar will then cause your pancreas to respond like a good watchdog and secrete the hormone insulin which will then lower your blood sugar back to the normal, healthy range. Easy enough, right? Well, fructose is different. If glucose could represent cars driving throughout the highway of your bloodstream, fructose would be like an ambulance roaring through the traffic, going around all the other cars until it arrives at its destination (the liver). Once in the liver, fructose has a front-of-the-line pass to be metabolized no matter how much was actually ingested. In biochemical terms, this type of event is described as "unregulated." What is the end result of this unregulated metabolism? The fructose that you eat puts an enormous strain on your liver by ravaging its energy supply in order for the fructose to be metabolized as quickly as possible, and this has serious health consequences.

In reality there are many diseases (pathologies) that can be attributed to the fact that fructose metabolism is unregulated. The effects of consuming large amounts of fructose through foods and drinks such as sugar-sweetened beverages are well linked to conditions such as non-alcoholic fatty liver disease (NAFLD), increased fat storage (making you fatter), decreased fat burning (keeping you fat), high blood pressure, muscle insulin resistance (impaired ability for muscle to lower your blood sugar when it is too high), kidney stones, and gout (painful buildup of uric acid in joints) -- just to name a few.^{7, 10-14} The increased cardiovascular risk from fructose consumption is evident in people of all ages too, especially in regards to adolescents and sugar-sweetened beverage consumption.¹⁵ In addition, there is evidence suggesting that over time large fructose consumption leads to an overall increased hunger level. This point about hunger seems counter-intuitive. Normally after we eat our hunger goes away, but fructose seems to contribute to chronic elevated insulin levels, which results in a continual state of hunger or a so-called state of *sugar addiction*.¹² One of the authors of the above journal review, a pediatrician and neuroendocrinologist named Robert Lustig, coined a memorable phrase, saying that fructose is like "alcohol without the buzz".¹⁶

If I have managed to convince you that fructose is dangerous, now you might ask the question, "How do I avoid fructose?" One of the most important things you can take away from this article is the fact that fructose makes up half of table sugar and over half of HFCS. Fruit and honey are the only major dietary sources of fructose found in nature, which is a very narrow scope, and based on the biochemistry, sources of dietary fructose should be kept to just that. Of course it is nearly impossible to eliminate sucrose altogether from our diets, but the fact is that your body is not well-equipped to handle the high levels of fructose that are found in many processed and packaged foods. Things like rice and potatoes are essentially giant LEGO® block creations of glucose, but not *any* fructose.

“
From a historical perspective fructose was virtually absent from our diet just a few hundred years ago.”
”

From a historical perspective fructose was virtually absent from our diet just a few hundred years ago.¹⁰ Until industrialization and the rise of corn production, the sources of fructose in our diet were limited to mainly honey and fruit. Please understand too that the amount of fructose in fruit is small compared to sugar-sweetened soda, for instance, and therefore should not be of much concern (Table 1).^{7,10} Many people ask "Why is HFCS used so much more than say, regular corn syrup or sucrose?" The answer has several parts, but what I want you to know is that fructose tastes *sweeter* than glucose. So, if a 50/50 mixture of glucose and fructose is converted to a new ratio of say 45% glucose and 55%

fructose, which is the standard in HFCS, the end product tastes better to most people. Clever, right? Sounds like more of a killer-filler to me.

It pays off tremendously to know exactly what your food is made of (for instance, that table sugar is 50% fructose), and I believe that having a conceptual understanding of why certain foods are hazardous to your health is crucial for making the necessary steps to achieve better dietary habits. If fructose is really as bad as it is thought to be, our public health campaigns should be targeted to all fructose-containing sugars.

REFERENCES

- [1] Beck-Nielsen H, Pedersen O, & Lindskov HO. 1980. Impaired cellular insulin binding and insulin sensitivity induced by high-fructose feeding in normal subjects. *American Journal of Clinical Nutrition*. 33, p. 273-278.
- [2] Hung CT. 1989. Effects of high-fructose (90%) corn syrup on plasma glucose, insulin, and C-peptide in non-insulin-dependent diabetes mellitus and normal subjects. *Journal of the Formosan Medical Association*. 88(9), p. 883-885.
- [3] Cohen, A. M., Teitelbaum, A., & Rosenman, E. 1977. Diabetes induced by a high fructose diet. *Metabolism*. 26(1), p. 17-24.
- [4] Tobey TA, Mondon CE., Zavaroni I, & Reaven GM. 1982. Mechanism of insulin resistance in fructose-fed rats. *Metabolism*. 31, p. 608-612.
- [5] Fukuda H, Iritani N, & Tanaka T. 1983. Effects of high-fructose diet on lipogenic enzymes and their substrate and effector levels in diabetic rats. *Journal of Nutritional Science and Vitaminology*. 29(6), p. 691-699.
- [6] Hwang IS, Ho H, Hoffman BB., & Reaven GM. 1987. Fructose-induced insulin resistance and hypertension in rats. *Hypertension*. 10, p. 512-516.
- [7] Tappy L & Lê KA. 2010. Metabolic effects of fructose and the worldwide increase in obesity. *Physiology Reviews*. 90(1), p. 23-46.
- [8] Wilt TJ, Shauck A, Shamliyan T, Taylor BC, MacDonald R, Tacklind J et al. 2010. *Lactose Intolerance and Health. Evidence report/Technology assessment*. 192, p. 1-410.
- [9] Basciano H, Lisa F, & Adeli K. 2005. Fructose, insulin resistance, and metabolic dyslipidemia. *Nutrition & Metabolism*. 2(5).
- [10] Bantle JP. 2009. Dietary fructose and metabolic syndrome and diabetes. *The Journal of Nutrition*. 139(6), p. 1263-1268.
- [11] Johnson RK et al. 2009. Dietary sugars intake and cardiovascular health. *Circulation*. 120, p. 1011-1020.
- [12] Lim JS, Mietus-Snyder M, Valente A, Schwarz JM, & Lustig RH. 2010. The role of fructose in the pathogenesis of NAFLD and the metabolic syndrome. *Nature reviews: Gastroenterology & hepatology*. 7(5), p.251-264.
- [13] Choi HK, Willett W, & Curhan G. 2010. Fructose-rich beverages and risk of gout in women. *Journal of the American Medical Association*. 304(20), p. 2270-2278.
- [14] Brown CM, Dulloo AG, Yepuri G, & Montani JP. 2007. Fructose ingestion acutely elevates blood pressure in healthy young humans. *American Journal of Physiology: Regulatory, integrative and comparative physiology*. 294(3), p.730-737.
- [15] Welsh JA, Sharma A, Cunningham SA, & Vos, MB. 2011. Consumption of added sugars and indicators of cardiovascular disease risk among US adolescents. *Circulation*. 123, p. 249-257.
- [16] Lustig RH. 2010. Fructose: metabolic, hedonic, and societal parallels with ethanol. *Journal of the American Dietetic Association*. 110, p.1307-1321.
- [17] USDA Agricultural Research Service. 2010. *Nutrient data laboratory*. Retrieved March 26, 2011, from <http://www.nal.usda.gov/fnic/foodcomp/search/>
- [18] The Coca-Cola Company. 2009. Nutrition connection. Retrieved March 26, 2011, from <http://productnutrition.thecoca-colacompany.com/welcome>
- [19] Pepsi-Cola Company 2011. Pepsi product information. Retrieved March 26, 2011, from <http://>